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LETTERS TO THE EDITOR

Effect of Smoking Status on Response to Thrombolytic Therapy

Barbash et al. (1) analyzed the relation between smoking status and outcome after thrombolytic therapy for acute myocardial infarction in the GUSTO-1 trial. The authors concluded that the superior survival of smokers is mostly accounted for by their younger age. However, in their analysis of the entire cohort, smoking remained an independent predictor of survival after age had been accounted for, which is in agreement with similar observations by the same authors using the international study data (2). It was only in their analysis of the angiographic substudy of GUSTO that smoking status was no longer predictive of survival after correction for age and gender. However, this may have resulted from inadequate correction for Thrombolysis in Myocardial Infarction (TIMI) flow grade.

We recently reported data from the TIMI-4 trial (3) showing that smokers more frequently have TIMI grade 3 flow in their infarct-related arteries 90 min after initiation of thrombolysis. This observation, previously reported by Gomez et al. (4) and now confirmed in the larger GUSTO data base, may explain in part the superior outcome of smokers after thrombolysis. Because improved early reperfusion may be a mechanism linking smoking and improved survival, it may be inappropriate to correct the observed mortality for the TIMI flow grade. We believe that younger age, as well as a better response to thrombolytic therapy, explain the superior outcome of smokers in acute myocardial infarction.

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References

1. Barbash GI, Reiner J, White HD, et al. Evaluation of paradoxic beneficial effects of smoking in patients receiving thrombolytic therapy for acute myocardial infarction: mechanism of the "smoker's paradox" from the GUSTO-1 trial, with angiographic insights. J Am Coll Cardiol 1995;26:1222–9.
2. Barbash GI, White HD, Modan M, et al. Significance of smoking in patients receiving thrombolytic therapy for acute myocardial infarction. Experience gleaned from the International Tissue Plasminogen Activator/Streptokinase Mortality Trial. Circulation 1993;87:53–8.
3. Zahger D, Cercek B, Cannon CP, et al. for the TIMI-4 Investigators. How do smokers differ from non-smokers in their response to thrombolysis? Results from the TIMI 4 trial. Am J Cardiol 1995;75:232–6.
4. Gomez MA, Karagounis LA, Allen A, et al. Effect of cigarette smoking on coronary patency after thrombolytic therapy for myocardial infarction. Am J Cardiol 1993;72:373–8.

Reply

We appreciate the comments made by Zahger and Shah regarding the effect of smoking status in response to thrombolytic therapy.

Smokers have numerous favorable clinical as well as angiographic characteristics compared with nonsmokers. The multivariable logistic regression helps us to define the correlation among these variables. Although age clearly plays an important role, we agree that the better outcome in smokers may well be related to the observed higher rate of Thrombolysis in Myocardial Infarction grade 3 flow in this group as well as overall less extensive coronary disease. The mechanism for the apparent better response to thrombolysis remains unclear and, as stated in the report, may derive from a different underlying pathogenic mechanism of the coronary lesions in these patients.

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Prognostic Value of Coronary Calcification—I

Inexplicably, Detrano et al. (1) have excluded coronary revascularization procedures from their analysis of coronary events in 491 symptomatic adults undergoing coronary arteriography for clinical indications. This omission is serious, for it assumes that coronary revascularization procedures fail to prevent nonfatal myocardial infarction or coronary death. The decision to exclude coronary revascularization procedures also raises a series of questions:

1. How were procedure-related deaths and myocardial infarctions analyzed?
2. Did any patients undergo either coronary angioplasty or bypass surgery between the time of coronary arteriography and electron beam computed tomographic (CT) scanning?
3. Was this a study of patients undergoing elective cardiac catheterization, or were patients admitted to the hospital for unstable angina, acute myocardial infarction or congestive heart failure also included in the study?
4. How do angiography and electron beam CT compare when revascularization procedures are included?

A statement in the methods section ("scan results did not generally influence the decision to perform coronary angiography") raises two other questions related to the appropriateness of the decision to exclude coronary revascularization procedures from the analysis:

1. How often and under what circumstances did scan results influence the decision to perform coronary angiography?
2. Given the novelty of electron beam CT scanning for the diagnosis of coronary disease and the paucity of prognostic information avail-

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